

This listing of claims will replace all prior versions, and listings of claims in the application:

Listing of Claims:

1-24. (canceled)

1 ~~25~~. (previously presented) A method for identifying a modulator of the binding of CCX CKR polypeptide to a chemokine comprising

(a) contacting an isolated or recombinant CCX CKR polypeptide having the amino acid sequence as set forth in SEQ ID NO:2, or a fragment or variant thereof, and the chemokine in the presence of a test compound, and

(b) comparing the level of binding of the chemokine and the polypeptide in (a) with the level of binding in the absence of the test compound, wherein

the CCX CKR polypeptide, fragment or variant can bind the chemokine in the absence of test compound and the variant has at least 90% sequence identity to SEQ ID NO:2,

the chemokine is selected from the group consisting of ELC (EBI-1-ligand chemokine), SLC (secondary lymphoid organ chemokine), TECK (thymus expressed chemokine), BLC (B-lymphocyte chemoattractant), CTACK (cutaneous T cell attracting chemokine), mMIP-1 γ (murine macrophage inflammatory protein 1 γ) and vMIPII (viral macrophage inflammatory protein II), and

a decrease in binding indicates that the test compound is an inhibitor of binding and an increase in binding indicates that the test compound is an enhancer of binding.

26. (canceled)

2 ~~27~~. (previously presented) The method of claim ~~25~~¹, wherein said contacting comprises contacting a cell expressing the polypeptide, fragment or variant.

28-36. (canceled)

3 ~~37~~. (previously presented) The method of claim ~~25~~¹, wherein the chemokine is labeled.

4 ~~38~~. (previously presented) The method of claim ~~37~~³, wherein the label is selected from the group consisting of a fluorophore, a chemiluminescent agent, an isotope label, and an enzyme or a combination thereof.

5 ~~39~~. (previously presented) The method of claim ~~25~~¹, wherein the test compound is labeled.

6 ~~40~~. (previously presented) The method of claim ~~39~~⁵, wherein the label is selected from the group consisting of a fluorophore, a chemiluminescent agent, an isotope label, and an enzyme or a combination thereof.

7 ~~41~~. (previously presented) The method of claim ~~25~~¹, wherein the CCX CKR polypeptide, fragment or variant is part of a cell fraction.

42. (canceled)

8 ~~43~~. (previously presented) The method of claim ~~25~~¹, wherein the chemokine is ELC.

9 ~~44~~. (previously presented) The method of claim ~~25~~¹, wherein the chemokine is SLC.

10 ~~45~~. (previously presented) The method of claim ~~25~~¹, wherein the chemokine is TECK.

11 ~~46~~. (previously presented) The method of claim ~~25~~¹, wherein the chemokine is BLC.

12 ~~47~~. (previously presented) The method of claim ~~25~~¹, wherein the chemokine is CTACK.

¹³~~48~~. (previously presented) The method of claim ¹~~25~~, wherein the chemokine is mMIP-1 γ .

¹⁴~~49~~. (previously presented) The method of claim ¹~~25~~, wherein the chemokine is vMIPII.

¹⁵~~50~~. (previously presented) The method of claim ¹~~25~~, wherein the variant has at least 95% sequence identity to SEQ ID NO:2.

¹⁶~~51~~. (previously presented) The method of claim ¹⁵~~50~~, wherein the variant has at least 98% sequence identity to SEQ ID NO:2.

¹⁷~~52~~. (new) The method of claim ¹⁶~~51~~, wherein the CCX CKR polypeptide has the amino acid sequence of SEQ ID NO:2.

Brief Description Of The Figures

Sub a2
Figure 1 shows the nucleotide sequence for a human CCX CKR (SEQ. ID NO:1) and the predicted amino acid sequence of the human CCX CKR polypeptide (SEQ. ID NO:2).

Sub a3
Figure 2 shows the CCX CKR sequence aligned with those of other chemokine receptors, the expression pattern of CCX CKR RNA, and generation of a stable cell line expressing CCX CKR. Figure 2A shows sequence homology of the CCX CKR coding region with other chemokine receptors. Figure 2B shows cells and tissues expressing CCX CKR RNA, as analyzed by RT-PCR of cytoplasmic RNA from cultured primary cells and whole tissues from various organs as indicated. Figure 2C shows a population of transfected HEK-293 cells stably expressing CCX CKR protein containing an N-terminal Flag epitope, comparing intensity of anti-Flag mAb staining relative to wild type HEK293 cells.

Figure 3 shows the identification of CCX CKR ligands by adhesion to stalkokines. Figure 3A shows interrogation of immobilized stalkokine (SK) by HEK293-CCX CKR cells, where 'control' = background adhesion of HEK293-CCX CKR cells to wells containing no stalkokine (anchoring antibodies and media are present); ELC-stalkokine (SK) = strong adhesion of HEK293-CCX CKR cells to locations containing ELC-stalkokines immobilized via anchoring antibodies; ELC-SK + soluble ELC, soluble TECK, or soluble SLC = ablation of adhesion in the presence of excess concentrations of soluble recombinant 'native form' chemokines as shown; ELC-SK + soluble MCP-3 = no diminution in adhesion in the presence MCP-3 as representative of many non-competing chemokines. Wild type HEK293 cells showed no adhesion to any of the sites (not shown). Figure 3B shows the quantitation of adhesion of HEK293-CCX CKR cells to ELC-stalkokine in the absence and presence of soluble chemokines from a representative experiment. Figure 3C shows the results of homologous competition binding assay using radiolabeled ELC in the presence of increasing concentrations of cold ELC on either HEK293-CCX CKR cells (filled squares) or wild type HEK293 cells (open squares).

Figure 4 shows the ligand binding fingerprint of CCX CKR. ¹²⁵I-ELC against a

Figure 4A:
Figure 4-
Definition of CCX CKR protein binding activity, as indicated by using ¹²⁵I-ELC against a